Hypothesis Concerning Etiology of Abnormal Clotting Specific to Type A Blood

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Introduction

While it has been well-documented that those with type A blood have a dramatically increased risk of clotting from infections and vaccines, the etiology of this phenomenon unique to type A blood has not been established. The following is a hypothesis of this cause that could inform new research directions that address the problem.

Abstract

All proteins in the body have a property of chirality. Biocompatibility of absorbed nutrients and interactions between cells both rely upon complimentary chiralities for biological compatibility. Included under this aegis are the proteins found in cellular membranes that control what may enter or exit a cell, but which, in the case of the immune system, are also responsible for the capacity of an immune cell to "grab on to" another cell.

In the specific case of leukocytes and platelets, the protrusive length (into the extracellular space,) degree of helicity, and chiral direction of these proteins dictate their effectiveness in attacking foreign bodies or supporting clotting.

I would suggest that something about the membrane proteins of leukocytes and platelets in those with type A blood is the key difference that leads to this clotting disorder. Furthermore, I propose that this difference has to do with a greater malleability in helical severity which, when taken to its greatest extreme, creates proteins that start out twisting in one direction and finish by twisting in another. With twists in both directions, these proteins would be able to adhere to the proteins protroding from the cellular membranes of nearby cells of the same type, causing clotting even when it is inappropriate. A protein with sections that feature both chiralities would tend to stick to everything, profoundly leading to host cells clumping together with other cells of the same type.

The trigger for this twisting would have to be a chemical signal resulting from an active infection. This chemical signal would not seem to result in abnormal clotting in those with B, AB, or O-type blood, thus I conclude that there is a difference in the composition of these membrane proteins in those with type A blood that causes them to experience an increase in helical severity and dual chirality where the membrane proteins of leukocytes and platelets of other blood types are impervious to this distortive effect. Where the membrane proteins in leukocytes in those with other blood types is like a very fine hair or stubble, the proteins protruding from type-A leukocytes, when chemically signalled to brace for an infection, are more like fuzzy Velcro. Since Velcro twists and curls in many directions, that material will securely adhere to whatever sort of miniature hooks it encounters. These corrupted membrane proteins function along similar lines.

From an evolutionary perspective, the mutation that causes this abnormal clotting, given that it does not seem to enhance immunity, nor does it seem to improve clotting when clotting is actually needed (many with type A blood report that they have increased clotting times from minor lacerations,) the mutation that led to this type would seem to be an evolutionary misstep and simply the result of a chance mutation.

Conclusion

The only way to prevent this clotting disorder would be to reprogram a person's immune system to manufacture white cells and platelets which feature an alteration of that single characteristic. Whether this could be done without inducing a dangerous autoimmune reaction is unclear. If successful, such a therapy would prevent many cases of ischemia.